

Neurological Digest.

CURRENT ANATOMY, PHYSIOLOGY AND PATHOLOGICAL
ANATOMY OF THE NERVOUS SYSTEM.

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PATHOLOGICAL CHANGES FOUND IN A CASE OF ANTERIOR PLOIOMYELITIS.

St. John Budden, *Journal Mental Diseases*, Jan. 18, 1892. The patient was sixty-one years of age, intemperate and syphilitic. No family history of neurotic taint, except his mother was said to be paralytic some years before her death. In consequence of an injury to the spine nine years previous he had paraplegia, from which he never completely recovered. A severe injury to his left hand two years and nine months afterward was followed by atrophy of the hand and arm, and five years later this was followed by atrophy of the opposite side. There was at this time a failure of sight and a change in the voice. Physical examination showed absence of reflexes both superficial and deep in all the extremities, and muscular percussion gave scarcely perceptible moves. The limbs were greatly wasted, the upper more than the lower, many of the muscles seemed to have entirely disappeared, marked wrist-drop, classic appearance of the hand, absence of thenar and hypothenar eminences. The lower limbs were more wasted below the knees. Muscular power was almost completely absent and when he did move the limbs slightly, the movements were irregular and incoördinate, skin was shiny and covered with branny desquamation. Considerable anæsthesia of the limbs and trunk, and sensation was delayed from all parts. Analgesia in all parts of the body except the face. Patient had no control over the bladder or rectum, sense of smell absent, taste and hearing fairly acute, slight internal strabismus of right eye and diplopia. Some five years before his death he suffered from distressing fulgorant pains in the limbs and muscular startings, and toward the latter part of his illness these became less intense, but they never completely disappeared. Although the patient was in bed for

five years bed sores were never troublesome and healed up quickly when they did occur. The electrical reactions were those of degeneration.

Sections cut on the freezing microtome and stained with blue-black; the ganglionic cells in the motor areas of the cortex were swollen and globose; not distinctly outlined and evidences of granular degeneration. The changes in the pyramidal cell layer were of the same character, but less well marked, the principal features being the pallor and indefiniteness of both cells and nuclei. The first layer showed the slightest evidences of degeneration, and the spindle was very slightly changed. The gray matter of the remaining convolutions showed simply evidences incident to senility.

The spinal cord was much shrunk, especially in the lumbar segment; microscopically examined it showed that the entirety of the posterior columns was diseased, most marked at the periphery. All the nerve fibres were atrophied, but there was no perceptible increase of connective tissue here, and the degenerations seemed to be continued but a short distance into the lateral columns. The remaining white columns showed a crowding together and an indistinctness of detail. In the anterior cornua no healthy nerve cells could be found; they were tinted, shrivelled and much pigmented.

The dorsal segment showed similar changes in the posterior columns, except that a great number of vessel orifices was found here. The anterior columns in this region gave a distinct increase in connective tissue as also did the lateral columns just outside the anterior horns. The nerve fibres in the peripheral parts of the anterior zone were all very much wasted. In the crossed pyramidal tracts the changes were the same. Clarke's columns were scarcely discernible, and no cells could be found in the anterior cornua.

The disease was much less marked in the white matter of the cervical region, and decreased as the medulla was approached, the column of Goll being most affected at the periphery and the changes in the posterior root nerves remaining the same. The cell disintegration in the anterior horns was as in the other regions. The degeneration in the posterior columns continued into the medulla, but the nuclei of the funiculus gracilis and funiculus cuneatus seemed to be intact here. The only nucleus in the medulla which seemed to be diseased was that of the 12th nerve.

The changes in the peripheral nerve were those of parenchymatous atrophy, many of the nerve bundles having entirely disappeared, the larger ones showing a loss of contour and great shrinkage. These changes were most marked in the sciatic nerves. Portions of the muscles taken from the flexors and extensors all showed atrophy of the fibres with granular disintegration and infiltration of the areolar tissue with fat, the greater part of the muscle being composed of densely nucleated connective tissue. In the cauda equina the degeneration in the nerve fibres was very extensive, and the pia mater about the cord was thickened and very dense.

The author thinks the patient first suffered from an acute poliomyelitis which was only partially recovered from, and that the crushing of the hand acted as an exciting cause in the development of the disease in the upper extremities, and this, with the multiple neuritis, which was subsequently developed would account for the symptoms. It is also the opinion of the author that the peripheral neuritis was secondary to the cornual changes, as the atrophy of the limbs proceeded indiscriminately and independently of the sensory disturbances. As regards what part the changes in the posterior columns played in the development of the symptoms, he considers it impossible to say, on account of the date of the beginning of the anæsthesia, or whether it was preceded by hyperæsthesia not having been established. The writer does not try to account for the changes in the cortex, or the influence they had in the course or development of the symptoms, but it is to be noted that they were confined to the motor areas.

A CASE OF PROGRESSIVE MUSCULAR ATROPHY, WITH DISEASE OF THE NUCLEI OF THE BULB AND THE CORTEX.

Dr. A. Alzheimer (*Archiv. f. Psych.*, vol. xxiii, part ii, p. 459). The case reported is that of a merchant, 33 years of age, single and descended from a family without neuropathic history. In 1876 the patient suffered from an attack of typhoid fever; in 1877-78 an abscess in the femur troubled him, which resulted in a fistula, continuing till the time of his death. In 1879 there was first noticed a wasting of the thenar eminence. He became infected with syphilis in 1881, and was treated with mercury and no secondary symptoms were manifest. Toward Christmas of 1889 he suffered severely from pain in the stomach and vomiting, and in Feb-